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Non-invasive diagnosis and assessment of aortic valve disease and evaluation of aortic prosthesis function using echo pulsed Doppler velocimetry*

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SUMMARY Non-invasive recording of aortic blood flow velocity patterns in the ascending aorta and in the arch of the aorta was performed in 12 normal subjects, 38 patients with confirmed aortic valve disease, and 13 patients with aortic prostheses using pulse echo Doppler velocity recordings. In normal subjects, the velocity recordings correlated well with those obtained by other authors using invasive procedures. In patients with aortic valve disease, specific abnormalities of the velocity curves were found to correlate well both with the type of lesion (stenosis or regurgitation) and its severity on a three-point scale. Both sensitivity and specificity were found to range between 80 and 94 per cent. A less accurate grading of severity was obtained from patients with aortic regurgitation by the detection of turbulence in the left ventricular outflow tract than from the appearance of the aortic velocity curves. In the studies of patients with aortic prostheses, anomalies of the velocity pattern could be found in the ascending aorta in 53 per cent but no abnormalities of timing were found.

In spite of some technical limitations, pulse echo Doppler velocity recordings provide a new non-invasive, reliable, and reproducible approach in assessing the presence and severity of aortic lesions and demonstrating flow abnormalities produced by prostheses.

The accurate diagnosis and assessment of the severity of aortic valve disease as well as malfunction of aortic valve prostheses require invasive procedures. None of the non-invasive techniques such as phonocardiography, continuous wave Doppler arterial blood velocity measurements, or standard echocardiography can give a full evaluation of aortic valve function in all types of lesion. Two-dimensional echocardiography, though helpful in many lesions, is not sufficiently accurate in the assessment of aortic regurgitation and does not by itself provide useful information about flow dynamics of the aortic valve. The recent development of pulse echo Doppler blood velocity recordings by our group[1–3] enables the clinician to record directly and non-invasively the flow velocity patterns in the aorta and prompted us to find out whether it could provide a useful technique for the evaluation of aortic valve function.

Subjects and methods

The control group consisted of 12 healthy subjects (four female and eight male), ranging in age from 5 to 54 years (mean 24), in whom any suspicion of organic heart disease had been ruled out by standard non-invasive procedures. A group of 38 subjects with aortic valve disease was studied (11 female and 27 male) ranging in age from 13 to 77 years (mean 49), including 11 with pure aortic stenosis, 17 with pure regurgitation, and 10 with combined stenosis and regurgitation (Tables 1–3). The aetiology of the aortic stenosis was found to be rheumatic in six cases, congenital in five cases, and degenerative with calcification in 10 cases. The aetiology of the aortic regurgitation was rheumatic in 10 cases, endocarditis in two cases, aortic dissection in one case, congenital in one case, atheroma in one case, and paravalvular prosthetic leak in one case. There was also one case of Pezzi-Laubry syndrome.

Four of these cases had associated mitral valve lesions, and two had mitral prostheses; 31 were in sinus rhythm, five had atrial fibrillation, one ventricular paced rhythm, and one second degree atrioventricular block. Five patients had left bundle-branch block. The diagnosis and assessment of the severity of these lesions were confirmed in all cases, either by retrograde left heart catheterisation or at the time of surgery, or both. Twenty patients

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Table 1 *Haemodynamic and surgical data of patients with aortic stenosis*

Case no.	Rhythm	LV pressure (mmHg)		Aortic pressure (mmHg)		CI (l per min per m²)	Aortography	Volume of regurgitation % CO (DDC)	Aortic valve area (cm²) (catheterisation or surgery)
		S/D	ED	S/D	Mean				
1	SR	NP		120/70	90	2·4	AS		0·30
2	SR	NP		120/67	86	2·9	AS	<5	
3	SR	204/4	10	116/60	82	2·8	AS	<5	0·50
4	SR	NP		106/68	88	3·0	AS	<5	0·30
5	SR	NP		174/88	122	3·3	AS		0·15
6	SR	NP		120/60	80		AS	<5	1·0
7	SR	NP		110/66	84	1·5	AS		0·2
8	SR								
9	SR								2·25 (post-valvotomy)
10	AV block	114/0	4	100/60	78	2·6			1·55
11	PM	NP		135/52	88	1·9			1·0

SR, sinus rhythm; AV, atrioventricular; PM, pacemaker; LV, left ventricle; S/D, systolic/diastolic; ED, end-diastolic; CI, cardiac index; CO, cardiac output; DDC, dye dilution curves; MS, mitral stenosis; MR, mitral regurgitation; NP, left ventricle catheterisation not possible.

Table 2 *Haemodynamic and surgical data of patients with aortic regurgitation*

Case no.	Rhythm	LV pressure (mmHg)		Aortic pressure (mmHg)		CI (l per min per m²)	Aortography	Volume of regurgitation % CO (DDC)
		S/D	ED	S/D	Mean			
12	SR	80/10	25	80/50	65	2·1	+++	
13	AF							
14	SR	115/0	15	145/55	100	1·5	+++	
15	SR	200/0	5	200/140	90		+++	
16	SR	170/5	30	165/50	85		++	
17	SR	130/4	8	125/45	80	2·7	+++	
18	SR	132/0	26	130/82	45	1·7	++	31
19	SR	145/2	7	145/66	105	2·9	++	40
20	SR	200/0	8	200/72	125	2·8	++	30
21	SR							
22	AF	125/0	10	125/75	90		++	
23	AF	130/8	2	125/25	70	2·7	+++	
24	SR	170/6	26	170/90	122	2·4	+	15
25	SR							
26	SR	145/2	10	140/45	80	2·2	++	40
27	AF	115/0	10	115/45	80	1·9	++	
28	AF	120/2	4	105/55	80	2·1	+	20

AF, atrial fibrillation; VSD, ventricular septal defect; Mit. pr., mitral prosthesis; ASD, atrial septal defect. Other abbreviations as in Table 1.

Table 3 *Haemodynamic and surgical data of patients with associated aortic stenosis and regurgitation*

Case no.	Rhythm	LV pressure (mmHg)		Aortic pressure (mmHg)		CI (l per min per m²)	Aortography	Volume of regurgitation % CO (DDC)	Aortic valve area (cm²) (catheterisation or surgery)	Surgery
		S/D	ED	S/D	Mean					
29	SR									+
30	SR									+
31	SR	180/10	22	160/110	80	2·8	++		1·0	+
32	SR	112/0	5	100/55	65	2·3	+		1·0	+
33	SR	220/0	28	185/90	120		++		1·2	-
34	SR								0·9	+
35	SR	190/4	20	180/60	104	2·2	+++	45	1	+
36									0·4	+
37	SR	NP		130/64	90	2·5	++	35		+
38	SR	NP		114/56	80	2·3		17		+

CAS, coronary atherosclerosis. Other abbreviations as in Tables 1 and 2.

Surgery	*Aortic stenosis grading*	*Associated lesions*	*Aetiology*
+	3		Calcified degenerative
+	3		Calcified bicuspid valve
+	3		Calcified degenerative
+	3		Calcified degenerative
+	3	Renal insufficiency	Calcified dengenerative
+	2		Calcified degenerative
+	3		Calcified bicuspid valve
+	3		Calcified degenerative
− (previ. commis-surotomy)	1		Congenital stenosis, post-commissurotomy
−	1	MS+MR	Rheumatic
+	2	MS+MR	Calcified, rheumatic

Surgery	*Aortic regurgitation grading*	*Associated lesions*	*Aetiology*
+	3		Infective endocarditis
+	1	MS	Rheumatic
+	3	VSD	Pezzi-Laubry SD
+	3		Rheumatic
+	2		Rheumatic
+	3		Rheumatic
+	2		Rheumatic
+	2		Rheumatic
+	2		Rheumatic
+	3		Infective endocarditis
−	2		Rheumatic
−	3		Rheumatic
−	1	Cardiomyopathy	Degenerative
+	3	Mit. pr.	Prosthesis disinsertion
−	2		Dissecting aorta
+	2		Rheumatic
−	1	ASD	Congenital

Aortic stenosis grading	*Aortic regurgitation grading*	*Associated lesions*	*Aetiology*
2	1		Rheumatic calcified
2	1		Calcified degenerative
2	2	Mit. pr.	Rheumatic calcified
2	1	MS+MR	Rheumatic
2	2	CAS	Calcified degenerative
2	1		Bicuspid calcified valve
2	3		Bicuspid calcified valve
3	1		Calcified rheumatic
2	2		Calcified degenerative
2	1	CAS	Calcified degenerative

had retrograde left heart catheterisation using a Telco manometer, 26 cases had aortography and/or dye dilution curves with indocyanine green (13 cases) using the Waters apparatus, and 30 patients were assessed at the time of operation. Cardiac output was measured by the dye dilution technique. There was a third group of patients with aortic valve prostheses, ranging in age from 12 to 73 years (mean 40), of whom nine had a Starr-Edwards prosthesis, two a Björk-Shiley prosthesis, one a Lillehei-Kaster prosthesis, and one a Hancock prosthesis. Of these 13 patients (four female and nine male), four also had a mitral prosthesis. A fourth group of 13 subjects with heart disease not involving the aortic valve (eight female, five male) ranged in age from 8 to 62 years (mean 37); three had pulmonary stenosis, three had mitral stenosis, two had ventricular septal defects, and one had an atrial septal defect. One of these patients had isolated right bundle-branch block and one had arterial disease involving the lower limbs. There was also one with hypertrophic cardiomyopathy and one with Fallot's tetralogy.

CLASSIFICATION OF PATIENTS WITH AORTIC VALVULAR DISEASE

Three grades of severity were used: 1 (mild); 2 (moderate); 3 (severe). The classification of the pure or predominant lesion on this three-point scale was based partly on the symptoms and on the clinical presentation (bedside examination, chest *x*-ray, electrocardiogram, and clinical course) and partly on the haemodynamic and/or the surgical data. In the patients with aortic stenosis, the grading was based on the calculated aortic valve area assessed either by the findings at cardiac catheterisation using the Gorlin formula[4] (valve area greater than 1·5 cm^2 for grade 1, from 0·8 to 1·5 cm^2 for grade 2, and less than 0·8 cm^2 for grade 3), with correction for any aortic regurgitation proven by aortography or dye dilution. In some cases the left ventricular aortic gradient could not be measured as at the time of left heart catheterisation the aortic valve could not be crossed. In some instances when the patient was very ill, no cardiac catheterisation data were available as the patient went straight to surgery. In these cases, the degree of stenosis was evaluated from assessment of the appearance of the valve by the surgeon who took into account the severity of the commissural fusion and estimated the approximate area for the valve obtained from two-dimensional measurements. Four cases of tight calcific stenosis associated with insignificant regurgitation (less than 5% on the dye dilution curves) were considered as pure stenosis (cases 2, 3, 4, and 6). Aortic regurgitation was assessed by dye

dilution curves[5] (<20% of systemic output = grade 1; 20 to 40% = grade 2; >40% = grade 3) or by aortography (+ = slight regurgitation disappearing at the next systole; ++ = regurgitation with opacification of the ventricle still present at the next systole, with progressive opacification of the left ventricle; +++ = immediate total left ventricular opacification). Surgical findings were also taken into account whenever appropriate. The three-point scale aortographic classification was used to avoid possible overlap between the grades when using a four- or five-point scale.[6 7] This was to avoid a statistically significant risk of erroneous grading being introduced.[8] The same three-point grading was used for associated lesions. Since this group of patients studied included few mild lesions, 10 additional subjects, five with mild aortic stenosis and five with mild aortic regurgitation, were also studied to test the validity of the method. The diagnosis was assessed by other non-invasive procedures and on the clinical data including electrocardiography, chest *x*-ray, phonocardiograms, and echocardiograms. However, these additional cases were not included in the statistical analysis.

TECHNIQUE

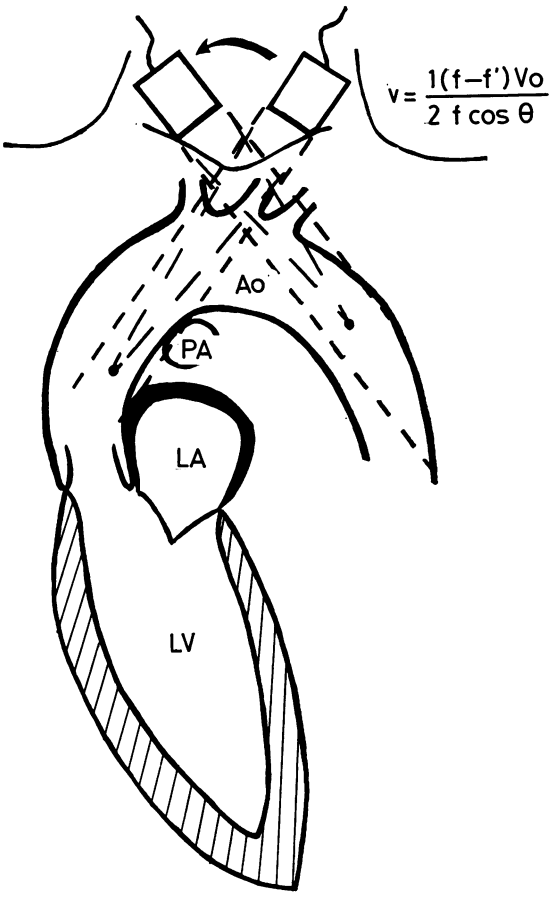
Range-gated ultrasonic pulsed Doppler blood velocity recording technique has been described elsewhere,[9 10] particularly the principle, apparatus, and the combined use of the Doppler technique with echocardiography using spectral analysis.[11]

(A) *Doppler principle*

The instantaneous flow velocity of a small (2 × 4 mm) teardrop-shaped sample volume of blood is proportional to the frequency shift between the ultrasonic beam emitted to, and the back-scattered ultrasound from, the red blood cells within the sample (Fig. 1). A range-gating system makes it possible to pick up any desired sample volume along the axis of the ultrasound beam within the limits of the equipment (3 to 17 cm).

(B) *Apparatus*

The ATL 500A blood flowmeter was used, combining a 3 MHz velocimeter which emits brief pulses (1·0 to 1·5 μs) at a pulse repetition of frequency of between 2 and 15 kHz (depending on the depths of the sample volume) and an echocardiograph having A- and M-mode. The echocardiograph and the Doppler recordings are made with a single transducer 1·2 cm in diameter. The output signal from the Doppler equipment is an audio signal which is fed to two loudspeakers and a spectral frequency display which consists of a time interval histogram. Additionally, our apparatus is equipped with an analogue velocity output obtained by frequency to voltage conversion of the audio signal using zero crossing detection technique. This equipment was used in our studies of the mitral valve.[2] The analogue velocity output can be



Transcutaneous aortic flow velocimetry (pulsed Doppler-echo)

Fig. 1 *The velocity of a small sample of blood is recorded at the same time as an echocardiogram which locates the position of the sample volume (echo shown by parallel dashed lines). The blood velocity is recorded according to the Doppler principle equation, upper right. Note the almost parallel orientation of the ultrasound beam and the long axis of the vessel minimising theta in the Doppler equation. The transducer is orientated downwards and is swept from the right anterior position for recording blood velocity in the ascending aorta to a left posterior position for recordings from the aortic arch. Depending on the depths of the range gate, several sample volumes may be obtained ranging along the ascending aorta and the arch. Ao, aorta; PA, right pulmonary artery; LA, left atrium; LV, left ventricle.*

recorded simultaneously or separately on a strip-chart recorder and demonstrates more obviously the anomalies which are also seen on the time interval histogram but are less easy to measure and evaluate on that type of display. The analogue velocity output represents a more convenient way of demonstrating cardiovascular correlation and is especially useful for grading the severity of lesions. The use of pulse echo techniques with limited pulse repetition frequency means that the maximum velocity that can be measured is limited and the true value of the flow velocity in the highly turbulent flow regimens seen in aortic valve disease is underestimated. The zero crossing technique to obtain an analogue blood velocity curve also results in underestimation of the flow velocity where there is a poor signal to noise ratio. It is therefore worth emphasising that the method described relies exclusively on pattern recognition and time anomalies in the flow velocity curves and not on the absolute value of velocity measured by this technique. The timing of the flow velocity characteristics was made from the analogue curve because they were easier to recognise there, but these measurements were always checked against the simultaneous time interval histogram spectrogram to make sure there was no distortion of the readout. The approximate time delay introduced by the frequency to voltage converter was approximately $30 \pm 0{\cdot}3$ ms when it was compared with the output from an electromagnetic flowmeter. Because of this delay in the velocity curve, the relative value and the absolute value of measurements were considered and compared with each other rather than made into absolute measurements. The ATL equipment includes a conventional echocardiograph with display of both A- and M-mode. A single transducer is used for all procedures; the location of the Doppler gate which controls the position of the sample volume is controlled by the operator and can be monitored on an oscilloscope. The device is linear to within 5 per cent from 0 to 100 cm/s for steady flow.

RECORDING TECHNIQUE

Echo-Doppler recordings include either simultaneously or separately the analogue velocity curve, spectral display, and M-mode echocardiogram showing the cardiac structures or major vessels. The recordings were made on an eight-channel photographic Irex recorder, together with lead II of the electrocardiogram, and a frequency selected phonocardiogram. Recordings were made at a chart speed of 50 or 75 mm/s, and the timing markers are at intervals of 40 ms. The tracings were recorded during apnoea or light respiration, with the patients lying flat on their backs initially with the head slightly extended and eventually with the shoulders raised on a pillow. Recordings were made most conveniently from the aorta with the transducer coated with conductive jelly placed in the suprasternal notch perpendicular to the skin so that the ultrasound beam successively passed through the aortic arch, the right pulmonary artery, and the left atrium[12] (Fig. 2). The transducer is then slightly angulated in a left posterior right anterior plane to superimpose both the axis of the ultrasound beam and the aorta. The Doppler gate is then adjusted to lie within the lumen of the vessel (Fig. 1). The correct orientation has been achieved when the typical Doppler audio signal is heard with minimal gain settings. It is possible by tilting the transducer and altering the depth of the sample volume to record several velocity wave forms at different sites within the ascending aorta and aortic root. The convention is followed of showing flow away from the left ventricle as a positive deflection and flow back towards the left ventricle as a negative deflection. This flow convention can be maintained by reversing a polarity switch when recordings are being made from the arch or descending aorta as opposed to the ascending aorta since the direction of flow with respect to the Doppler probe changes. Another approach to the ascending aorta is from the right supraclavicular fossa. From this site, the sample volume is set to depths from 5 to 8 cm. At 8 or 9 cm, the sample volume is very close to the sinuses of Valsalva. In that situation some of the aortic valve may be seen on the echocardiographic trace and a sharp sound may be superimposed on the Doppler audio signal if the valve enters the Doppler sample volume. A third approach is from the conventional echocardiographic access point along the left sternal edge. This approach is particularly useful for recording aortic valve motion velocities, turbulence, and prosthetic motion velocities, whereas aortic blood velocities are poorly recorded from this site as the ultrasound beam is almost at right-angles to the flow. A specific search for turbulence in the left ventricular outflow tract was always made in cases of pure aortic regurgitation to verify the site of regurgitation and prove that it was the aortic orifice.[11]

FEATURES OF VELOCITY RECORDINGS

Velocity patterns

In every recording from the ascending aorta and arch, we carefully studied: (1) the shape of the systolic "S" summit of the systolic curve; (2) the band width of the frequency spectrum; and (3) the pattern and polarity of the diastolic curve.

Timing

The following time intervals were measured on the velocity curves obtained from measurements from the ascending aorta and arch; RR intervals of the electrocardiogram; Q wave of the electrocardiogram to the onset of the systolic velocity curve (Q–O). The ejection time was determined from the analogue velocity (zero-crossing output) from the beginning to the end of the systolic wave. Three successive measurements were made from every subject and the average value thus obtained. All the time intervals were expressed in milliseconds and corrected for heart rate according to the Bazett formula.[13] In this series of 76 patients, from whom two basic recordings were made (total 152), 32 could not be used for timing measurements because of inadequacy of either the time interval histogram recording or the analogue signal. A statistical analysis of the results was performed using a Hewlett-Packard A851 computer used to derive mean values and standard deviations and also to perform Student's t test between the different groups of subjects.

Results

Timing data are summarised in Table 4.

The curves represent the blood velocity patterns of aortic blood flow at different points in the aorta. Since the output of the Doppler device is directional, negative flow, that is flow back towards the

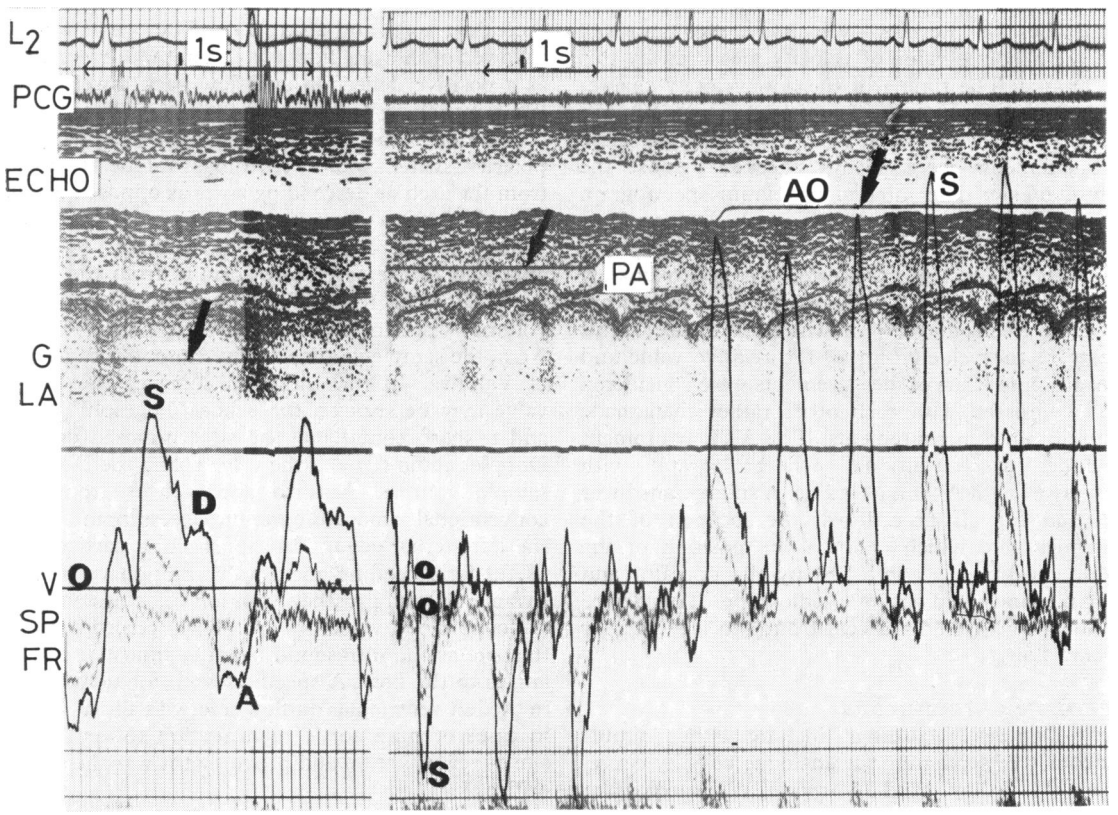


Fig. 2 *Suprasternal echo-Doppler scanning from the left atrium to the aorta in normal subjects. From above down, lead II ECG phonocardiogram (medium high frequency); echo with the sample volume position shown by "G" and at the bottom the analogue flow velocity output superimposed on the spectral display (FR). The sample volume position "G" is shown on the echocardiogram for successive recordings from the left atrium (LA) depth 9 cm: a high atrial velocity pattern is displayed on the left; next in the right pulmonary artery (PA) depth 7 cm: a negative systolic wave away from the transducer is recorded in the middle, and finally in the aorta (AO) depth 6 cm, a positive systolic wave towards the transducer of much higher amplitude is recorded here. A spectral negative S wave indicating aortic arch location of the sample is often shown using this orientation, but this is dependent upon the anatomy of the subjects.*

left ventricle is shown as a negative curve and flow away from the heart as a positive deflection.

NORMAL SUBJECTS (Group 1: Fig. 2, 3, and Table 4)

(1) *Ascending aorta*

The pattern of the spectral curve starts at the end of the first sound, and consists of a large positive triangular-shaped systolic wave, with a rapid upstroke, a sharp early peak labelled "S" followed by a less rapidly descending limb. It overrides the zero line and shows a precipitous, much smaller, negative deflection labelled the "R" wave. The velocity curve then rejoins the zero line and follows it throughout the remainder of diastole, showing a few small oscillations. The spectral output is made up of a thin regular coherent line of dots very closely spaced. The breadth of the spectrum is usually less than 1 cm, with discrete broadening of the spectrum at the top or on the descending limb of the summit "S". Small variations may occur consisting of a bifid pattern of the "S" wave or an irregular "S" summit amplitude, depending directly on the length of the preceding diastole in the case of a respiratory arrhythmia. When recordings have been made from close to

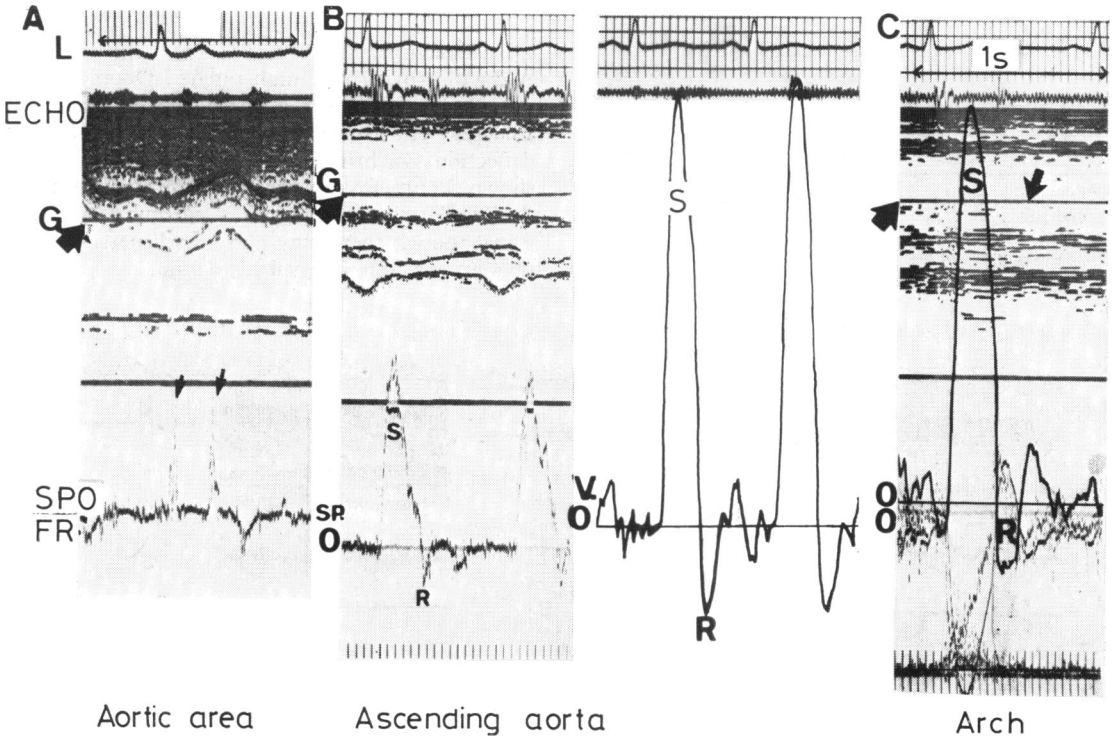


Fig. 3 *Normal subject.* (A) *Normal aortic orifice (from top to bottom: lead II of the electrocardiogram; ECHO echocardiogram; G, velocimetric gate with oblique arrows, SP, FR, frequency spectrum). The visualisation of the gate (horizontal line) in the centre of the aortic orifice documents the position of the Doppler signal. The spectrum frequency displays two thin and brief deflections (thin arrows), synchronous with the opening and closing of the aortic valves, generally having the same positive sign.* (B) *Normal flow velocity pattern in the ascending aorta (same legend as Fig. 2). With the gate (G) located in the aorta, a large systolic positive wave "S" is displayed on the spectral (left) as well as on the analogue (right) curve, documenting the location of the sample in the ascending aorta. Note the slower descending limb of the "S" summit corresponding to a slight physiological broadening of the spectrum, followed by a small brief reversal negative wave, "R". The amplitude of the "S" wave depends on the length of the preceding diastole.* (C) *From top to bottom: lead II of the ECG; PCG, phonocardiogram in medium high frequencies; SP, FR, spectrum frequency; V, analogue flow velocity curves with their respective zero. The gate is located in the aortic arch, as proven by the presence of a large negative systolic wave (flow velocity away from the transducer) on the spectral display: the pattern is similar to that seen in the ascending aorta. For the sake of a more physiological presentation, the convention is followed of showing flow away from the aortic valve as a positive deflection. The analogue curve is therefore reversed with respect to the spectral display.*

Table 4 *Aortic flow velocity timing data for normal subjects, patients with aortic valve disease, patients with aortic prosthesis, control group of cardiac patients*

Group		HR/min	Ascending aorta		Aortic arch	
			Q – O (ms)	ET (ms)	Q – O (ms)	ET (ms)
1	Normal subjects	79 ±16	130 ±25	328 ±33	136 ±17	318 ±25
2	A = AS	64 ±15	131 ±36	361 ±49	178 ±28	373 ±52
	p				< 0·01	< 0·02
AVD	B = AR	73 ±15	134 ±22	311 ±34	153 ±22	319 ±43
	C = AS + AR	68 ±9	138 ±29	352 ±45	175 ±15	342 ±53
	p				< 0·01	
3	Patients with Ao. pr	76 ±12	137 ±23	312 ±46	142 ±16	324 ±32
4	CCG (without AVD)	71 ±19	136 ±24	317 ±37	153 ±27	312 ±31

AVD, aortic valve disease; AS, aortic stenosis; AR, aortic regurgitation; Ao. pr, aortic prosthesis; CCG, cardiac control group; HR, heart rate; Q – O, Q to onset of the velocity curve time interval; ET, ejection time; ms, millisecond; p, p value; only the values of p under 0·05 are indicated in this Table.

the aortic valve, a negative diastolic wave is usually recorded but this disappears further away from the valve in the higher ascending aorta. The analogue velocity output shows the same triangular-shaped positive wave with broadly similar characteristics to the spectral display. The corresponding Doppler audio signal heard through the loudspeakers is short, exclusively systolic, and soft.

(2) *Arch*

The patterns are similar to those of the ascending aorta. Since the aortic flow is moving away from the transducer, the systolic deflection of the spectral analysis is negative (the polarity has not been reversed).

(3) *Aortic orifice*

A double sharp brief high-pitched Doppler sound may be heard in the loudspeaker, and on both the analogue and spectral velocity displays a short deflection synchronous with the cardiac sounds is shown. Between these two deflections, often having the same polarity, there is a narrow band width signal though there may be some dispersion of the dots on the time interval histogram.

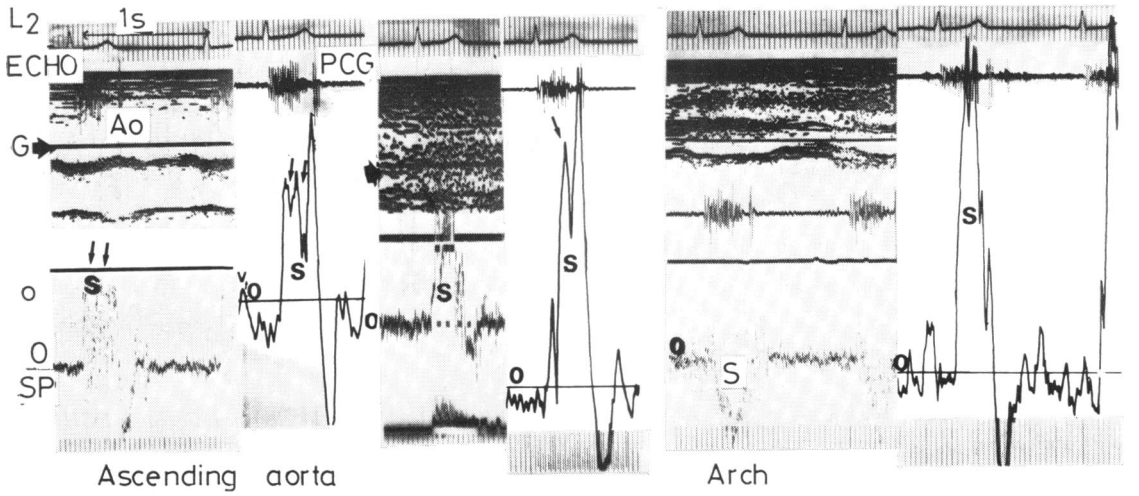


Fig. 4 *Mild aortic valvular stenosis (case 9, postoperative examination of a patient who underwent valvotomy for congenital aortic stenosis with a postoperative aortic area of 2·25 cm²) (same legend as Fig. 3A and B).*
Left: *velocity records in the ascending aorta as shown by the gate (G) and by the positive "S" wave on the spectrum frequency: both the analogue and the spectral curves show characteristic systolic anomalies consisting of the occurrence of coarse and important irregularities (arrows) replacing the normal triangular-shaped "S" wave, when the sample is near the aortic orifice; at a distance along the upper part of the ascending aorta (middle), the "S" summit progressively reappears with a slow and somehow irregular ascent, both on the spectrum and analogue.*
Right: *the gate is in the arch (negative systolic wave on the spectrum). The analogue curve is nearly normal, with a normal upstroke of the "S" wave. However, discrete irregularities are seen on its descending limb, as well as a discrete broadening of the corresponding part of the spectrum. The time delay between the Q-onset of the velocity curves in the ascending aorta and in the arch equals 0·04 s.*

PATIENTS WITH AORTIC VALVE DISEASE
(Group 2: Tables 1–4)

Aortic stenosis (Group 2A: 11 patients, Fig. 4 and 5)
Pattern and timing anomalies were found but only the former are described here.

(1) *Ascending aorta*
The normal triangular systolic pattern is not recorded but is replaced on the spectral display by a wide band width, a signal with considerable dispersion of the dots throughout most, or all, of systole. A slow ascent may be observed on the analogue output. Corresponding anomalies are that the "S" summit is amputated and replaced by an irregular saw-toothed plateau and on the loudspeakers, a loud, harsh prolonged sound is heard. These characteristic anomalies are usually recorded close to the aortic valve and spread a variable distance along the aorta.

(2) *Arch*
The systolic pattern may be very similar to that recorded in the ascending aorta consisting on the time interval histogram of a broadened spectrum and on the analogue display of an amputated saw-toothed summit, or the normal triangular-shaped systolic wave may reappear after a slower than normal ascent. The duration of the systolic wave is usually increased and the tracings frequently overlap in diastole.

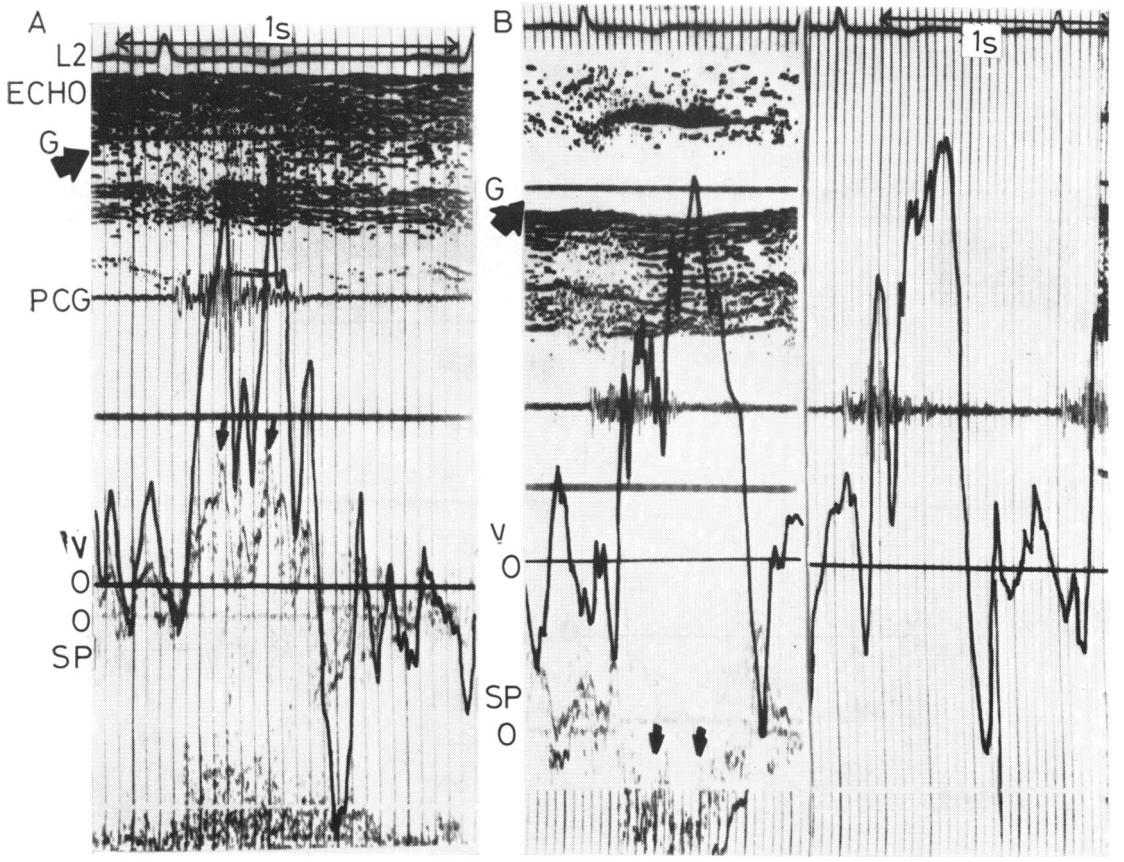


Fig. 5 *Aortic flow velocity pattern in pure and severe aortic stenosis (case 1—aortic orifice of 0·3 cm² at operation) (same legend as Fig. 2 and 3C).*
(A) *The gate (G) is in the ascending aorta. The normal summit "S" is cut off and replaced by the same coarse indentations as seen in the ascending aorta in the preceding case and a large broadening of the spectrum (depth recording 8 cm).* (B) *The gate is in the aortic arch, as proven by the negative systolic wave displayed on the spectrum: the same abnormal pattern is recorded in systole, with no reappearance of the "S" summit. Time delay between the Q-onset of the velocity curves in the ascending aorta and in the aortic arch equals 0·06 s.*

(3) *Aortic orifice*
The loudspeaker sound is loud and harsh in systole. The time interval histogram spectral display shows a wide rectangular-shaped dispersion of the dots spreading all over systole which is synchronous with the external murmur recorded on the phonocardiogram, where the brief initial and final deflections may be unrecognisable (see Fig. 9).

Severe stenosis (seven patients)
In these cases the abnormality found in the ascending aorta seems to spread further into the arch and is seen on both the spectral display and the analogue readout. There is delay in appearance of the systolic wave in the arch of the aorta; spectral broadening and indentations in the analogue velocity output occur very early in the ascending limb. Some of the curves show pronounced diastolic overlap with severe prolongation of the ejection time in four cases.

Mild stenosis (two proven cases and five non-invasively diagnosed cases), and
Moderate stenosis (two cases)
In all these cases except two (case 10 and one externally diagnosed), the characteristic features were present on the velocity curves recorded close to the aortic valve with a delay in the onset of the velocity curves in the aortic arch. However, the velocity anomalies progressively disappear with distance from the aortic valve, as can be seen from recording in the ascending aorta and in the arch (Fig. 4).

REGURGITATION (Group 2B: 17 patients, Fig. 6, 7, 8)
The characteristic anomaly consists of reversed flow in diastole shown as a reversed deflection on both the spectral and analogue curves.

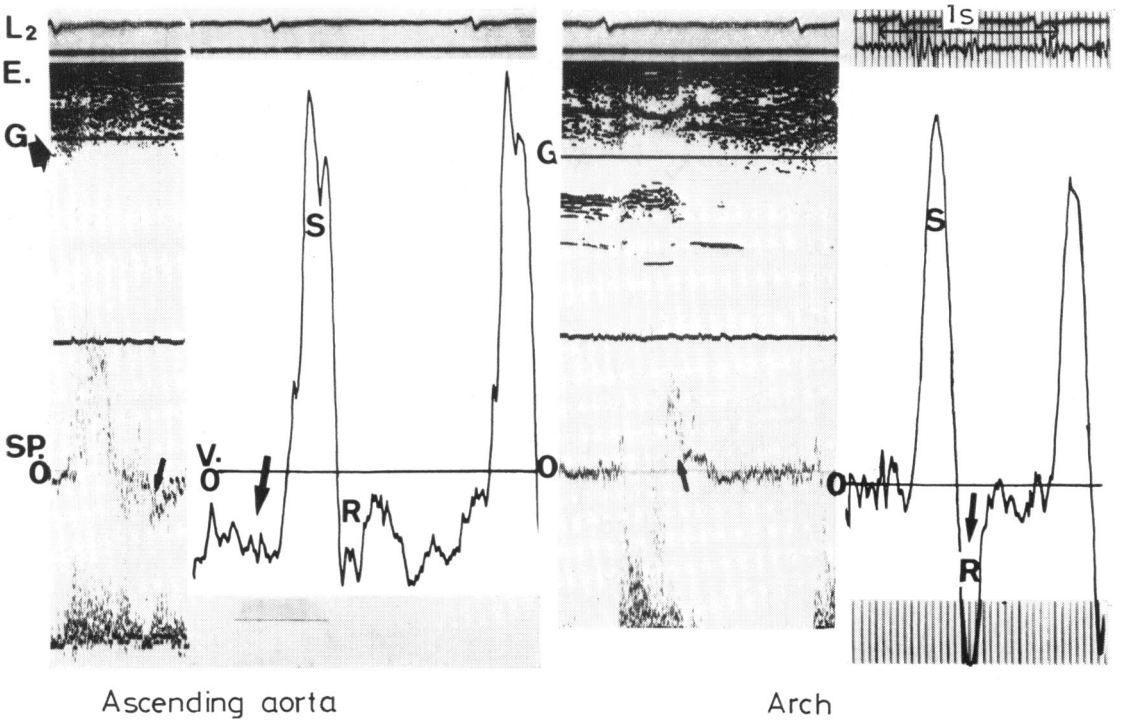


Fig. 6 *Aortic flow velocity patterns in pure and mild aortic regurgitation (case 24—same legend as Fig. 3B).*
Left: *in the ascending aorta, there is an abnormal negativity (oblique arrow) after the initial reversal wave, both on the spectrum and analogue.*
Right: *in the aortic arch, the diastolic pattern is nearly normal, except for a slightly wider reversal wave, followed by a discrete negativity on the analogue. There is no abnormal delay between the respective onsets of both curves.*

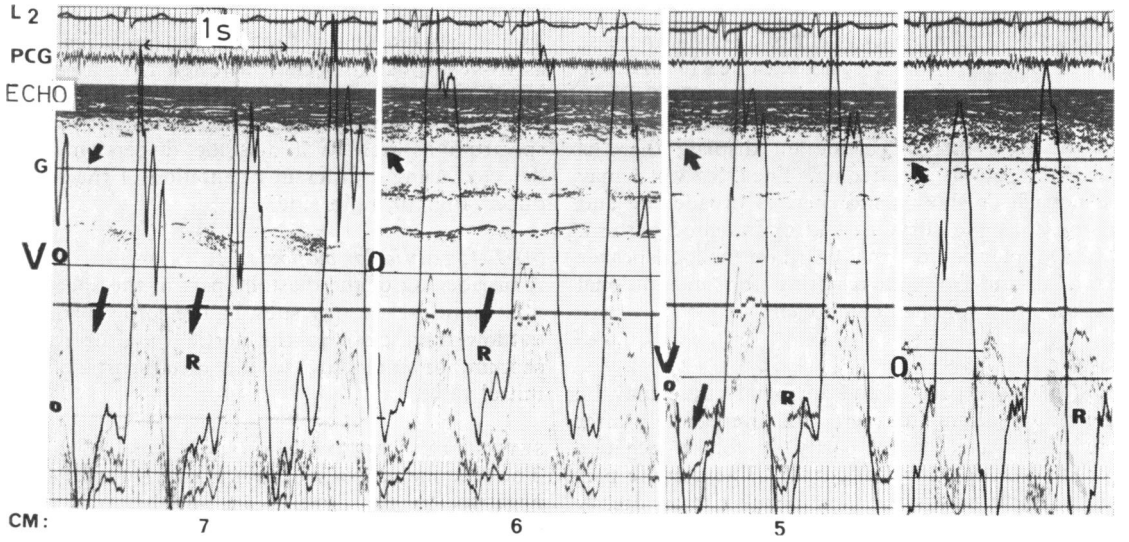


Fig. 7 *Aortic flow velocity patterns in pure and moderate aortic regurgitation (case 20, same legend as Fig. 2). The numbers below the traces indicate the depths of the sample volume from the skin. At 7, 6, and 5 cm velocity traces are recorded in the ascending aorta as the gate is moved progressively downstream from the aortic valve. The diastolic negative velocity deflection becomes progressively less pronounced. The zero calibration is shown in all the traces and moves downwards from left to right. The right-hand trace obtained in the aortic arch has much less reverse flow in diastole on the analgue velocity curve. Though the peak of the systolic curves show some irregular indentations, organic aortic stenosis can be ruled out by the timing of the onset of the systolic velocity wave S.*

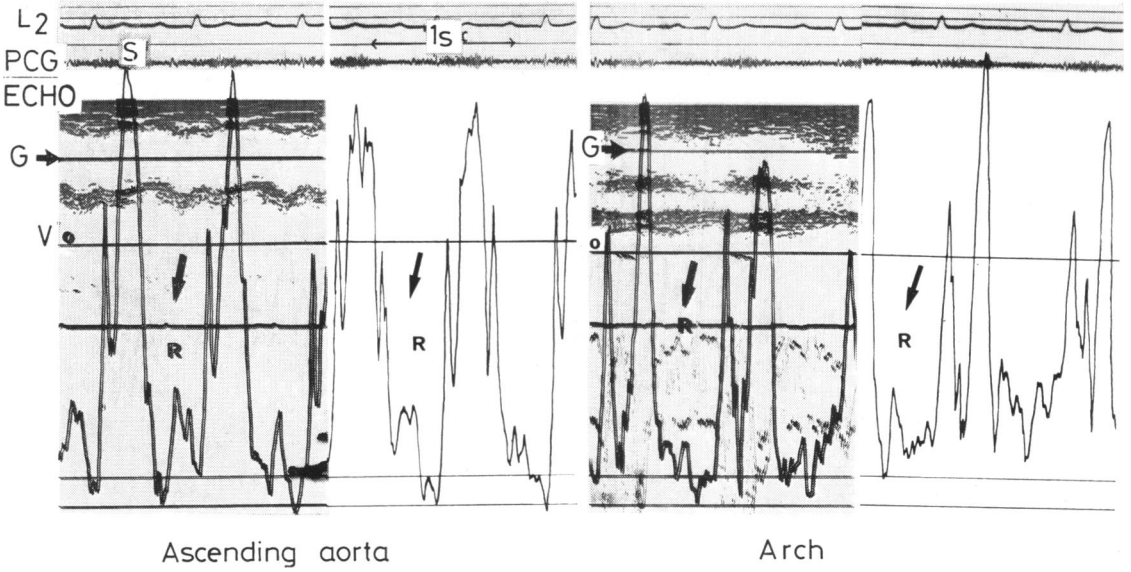


Fig. 8 *Aortic flow velocity patterns in pure and major aortic regurgitation (case 14—same legend as Fig. 2 and 3B).* Left: *ascending aorta. Note the conspicuously pan-diastolic negative wave ("R" oblique arrows) whose amplitude exceeds that of the systolic wave. The irregular pattern of the "S" wave is often seen in major regurgitation.* Right: *in the arch. There is still a distinct diastolic negative wave of considerable amplitude and duration. The bifid systolic pattern could be a result of an artefact, but is consistent with the classical systolic pattern seen in aortic regurgitation. No abnormal delay on the Q-onset interval.*

(1) *Ascending aorta*
In diastole, the anomaly is either a simple increase in the width of the physiological early diastolic reverse flow wave or a conspicuous negative wave of variable depth occurring for part or all of diastole. In the time interval histogram display, there is variable spectral broadening. The "S" wave may be normal or show some spectral broadening and in the analogue curve a bifid or indented pattern. The Doppler audio signal heard on the loudspeaker has a "to-and-fro" tone resulting from an additional diastolic high-pitched component.

(2) *Arch*
The diastolic part of the analogue curve may be normal, rejoining the zero line immediately after the "R" wave, or be partly or totally negative (positive on the spectral curve). The systolic part of the curve shows the same peculiarity as already described for the ascending aorta.

(3) *Aortic orifice*
The initial and final deflections are easily recorded as from normal subjects, but often have a greater amplitude with a sharper tone in the loudspeaker. There may be systolic dispersion of the dots on the spectrum as well as a diastolic dispersion often detected using a different orientation of the transducer (Fig. 10, right side).

(4) *Left ventricular outflow trace*
A broadening of the diastolic part of the spectrum was found at various sites in the left ventricular outflow tract close to the aortic valve or to the septum, or extending to the distal parts of the mitral valve.

SEVERE REGURGITATION (seven patients)
The diastolic part of the curve is entirely and deeply negative, as far downstream as the arch. The systolic wave shows indentations up to the arch;

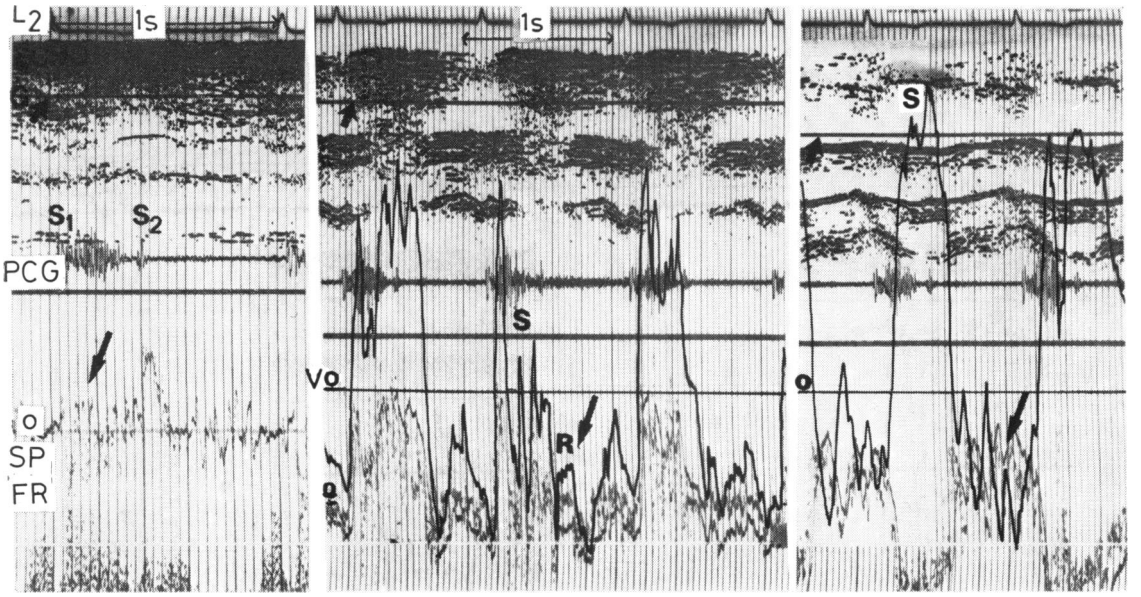


Fig. 9 *aortic orifice. Aortic flow velocity patterns in moderate stenosis associated with severe regurgitation (case 35 bicuspid calcified valve, same legend as Fig. 1 and 3A).*
Left: *aortic orifice. There is considerable dispersion of the dots in the spectral display (oblique arrow) indicating velocity disturbances; note the synchronous timing of the praecordial murmur on the phonocardiogram, with the dispersion of the dots on the spectral display. These disturbances are recorded only on intercostal space higher than the usual echo location of the transducer for examination of the aortic root and were only displayed with an asymmetrical location of the transducer disappearing when the centre of the aortic orifice was being sampled.*
Middle: *ascending aorta. Note the association of the characteristic abnormal systolic wave "S" with the negative wave throughout diastole ("R") (oblique arrow).*
Right: *aortic arch. The curve is delayed (delay of the Q-onset time interval equal to 0·05 s) and shows the same anomalies, though less pronounced in systole.*

the ejection time is sometimes prolonged. But the curve does not override the second sound and there is no significant time lag between the onset of the curves recorded at different levels of the aorta.

MODERATE REGURGITATION (seven patients)
The diastolic negativity is often important in the ascending aorta, but is much less obvious in the arch, though still present. The systolic wave shows a bifid pattern.

MILD REGURGITATION (three patients, and five additional externally diagnosed patients)
In all but three patients (case 13 and two non-invasively investigated patients), diastolic anomalies were elicited, consisting of negative phases of short duration, disappearing in the upper part of the ascending aorta, with a normal pattern at the arch. The systolic wave may be bifid or normal.

ASSOCIATED AORTIC STENOSIS AND REGURGITATION (Group 2C: 10 patients, Fig. 9)
Although not always present simultaneously, the analogue and spectral velocity curves show the same anomalies described in both the ascending aorta and arch for the separate lesions.

(1) *Moderate stenosis with mild regurgitation* (five patients)
The velocity curve shows systolic indentations in recordings made downstream from the aortic valve as far as the arch. The diastolic negativity disappears before the aortic arch is reached.

(2) *Moderate stenosis with moderate regurgitation* (three patients)
The same pattern is present for the systolic wave, but the diastolic negative flow velocity persists as far as the aortic arch though is less pronounced than in the ascending aorta.

(3) *Moderate stenosis with severe regurgitation* (one patient)
In a recording of the aortic arch, the systolic wave has the characteristic indentations with delay of onset of the curve and the diastolic part of the curve is conspicuously negative.

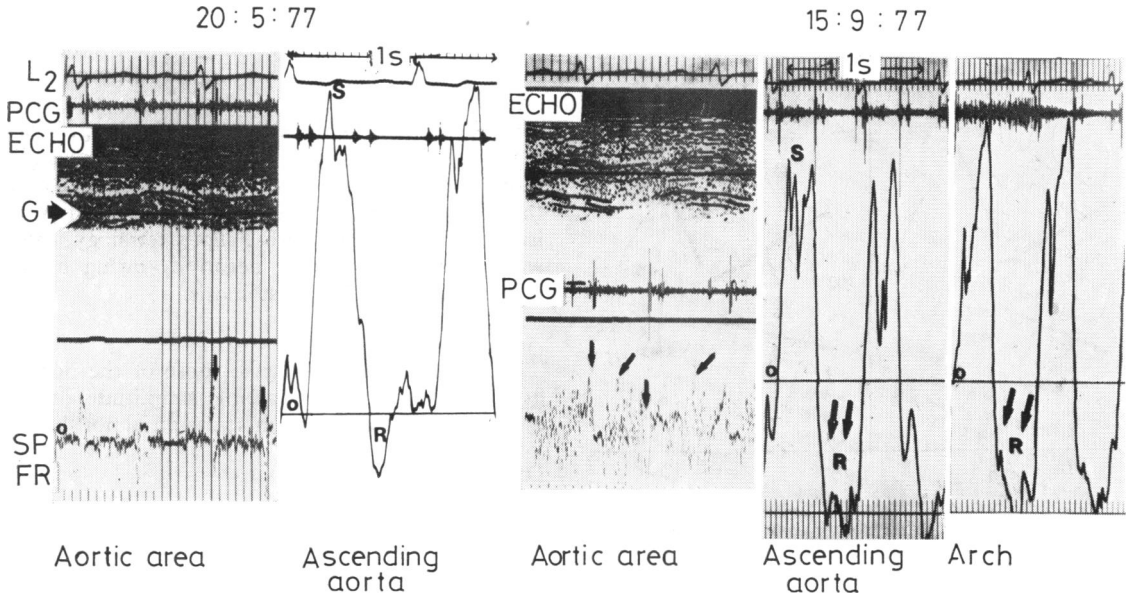


Fig. 10 *Aortic Björk prosthesis. Normal function (May 1977) (same legend as Fig. 3A and C). Praecordial recording of the velocity of prosthetic motion. The gate (G) is located at the level of the prosthesis and the record shows two very transient episodes of dot dispersion synchronous with the opening and closing of the prosthesis. There is also a narrow bandwidth of the spectrum in systole and diastole, indicating that there are no flow disturbances at the aortic orifice. The analogue flow velocity pattern is nearly normal and in the right-hand panel recorded several months later, the gate is located at the same position. Note the wide dispersion of the dots on the spectral display indicating velocity disturbances in systole and diastole. The analogue velocity curve also shows a bifid systolic pattern and pronounced diastolic negative flow indicating aortic regurgitation. At operation there was a paravalvular leak 1 cm² in area (case 25).*

(4) *Severe stenosis with mild regurgitation* (one patient)
In this case there was a typical stenotic pattern of blood velocity in the ascending aorta as well as diastolic negative flow typical of regurgitation, but the evaluation of the stenosis was unreliable, a single recording only being available.

PATIENTS WITH AORTIC PROSTHESES (Group 3: 13 patients, Fig. 10, left side)
(1) *Ascending aorta*
Transient dispersion of the dots is seen on the spectrum in more than half the cases. The corresponding analogue pattern is variable, being normal in four cases and bifid in two cases, with an indented "S" summit in seven cases. In four cases, slight diastolic negative velocity was present.

(2) *Arch*
The pattern shows some minimal disturbances of the spectrum and corresponding indentations on the summit of the S wave in the analogue curves (three patients). The Doppler loudspeaker signal is generally normal at the arch, and high-pitched but not prolonged in the ascending aorta.

(3) *Aortic orifice*
In all cases, two brief narrow deflections with opposite polarity were recorded, one at the beginning and the other at the end of systole. The respective polarities of the deflections were reversed using the suprasternal supraclavicular or thoracic approach. Between these two deflections, the velocity curve stayed close to the zero line. The loudspeaker signal had a sharp high-pitched tone.

CARDIAC PATIENTS WITHOUT AORTIC VALVE DISEASE (Group 4: 13 patients)
The anomalous summit pattern was found in two cases of pulmonary stenosis, one of them with Fallot's tetralogy. The other patients had normal systolic patterns in all aortic samples. A thin pattern with reduced systolic ejection time was found in one case of severe mitral regurgitation. In patients with atrial fibrillation the amplitude of the "S" summit varied depending on the previous diastolic interval. Where there was a long diastolic interval because of bradycardia or atrial fibrillation, the diastolic portion of the velocity curve may be irregular. In patients with Fallot's tetralogy, there was an obvious diastolic negativity owing to the presence of Blalock's anastomosis.

Aortic orifice
The findings were similar to those of the normal group except for the variable amplitude of the systolic deflections in patients with atrial fibrillation.

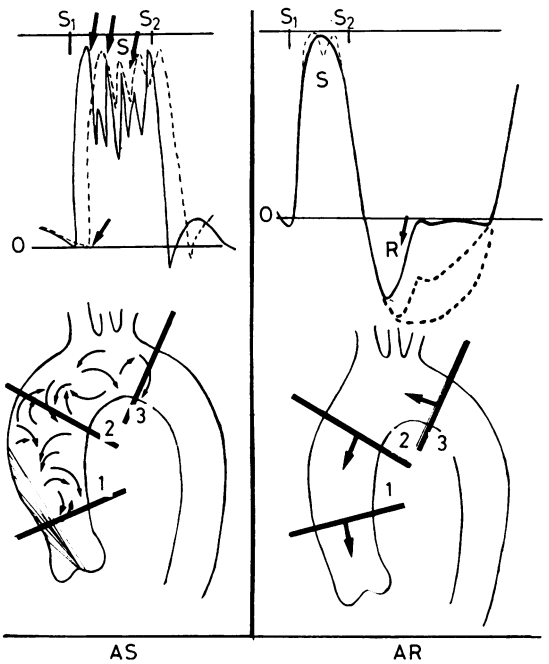


Fig. 11 *Schematic representation of the aortic flow velocity patterns in patients with aortic valve disease (grades 1, 2, and 3 correspond to mild, moderate, and severe lesion). AS, aortic stenosis; AR, aortic regurgitation; S1 and S2, first and second sounds.*
Left: *aortic stenosis. The typical indented systolic wave seen in the upper part may be recorded at various levels along the aorta, according to the severity of the lesion. The dotted curve represents the delayed velocity tracing recorded at the arch.*
Right: *aortic regurgitation. The depth and duration of the diastolic negativity of the velocity curve as well as the persistence of these anomalies lower down the aorta is related to the severity of the lesion.*

Discussion

Accurate diagnosis and evaluation of the severity of aortic valve disease by purely non-invasive methods has not so far been satisfactorily achieved. M-mode echocardiography as a rule provides no direct information about aortic valve lesions, but only indirect evidence based on its effect on left ventricular cavity size and wall thickness. Many uncertainties remain in assessing the severity of aortic valve disease and prosthetic valve function.[14] Cross-sectional echocardiography has the advantage of directly visualising the valve.[15] However, the accuracy of the diagnosis based on this technique

remains to be demonstrated for all types of aortic valve disease. These problems emphasise the need for a more direct approach based on direct demonstration of the flow disturbances produced by the aortic valve disease.

Non-invasive measurement of blood flow velocity from the aorta using the suprasternal notch has been performed in the last decade using continuous wave Doppler equipment.[16–18] However, such methods are not useful in all types of disease and have emphasised the need for a more precise technique which can measure the velocity from a more limited sample of blood in the aorta. This has been met by the advent of pulse echo Doppler techniques introduced by Johnson *et al.* in 1973.[11] Several papers have been published describing pulse Doppler flow velocity measurements from the aorta[3 19] with or without echocardiography.[20] More recently, cross-sectional echocardiography has been combined with pulse Doppler techniques[21] but routine aortic velocity recordings could not be obtained. The method described here is based on velocity recordings from several different sites at various points downstream from the aortic valve, viz at the orifice in the ascending aorta, in the aortic arch, and in the proximal descending aorta. Diagnosis and assessment of the severity of the aortic valve lesions rely on pattern recognition and the timing of velocity events obtained from these curves. The main problems raised by the use of continuous wave Doppler blood velocity measurement have been discussed at length in previous publications.[22 23] It is pertinent to mention some technical limitations raised by the present equipment which is based on pulse echo Doppler, the time interval histogram, and zero-crossing rate meter. It is well known that the output of a zero-crossing meter, whether fed to an analogue velocity curve or a time interval histogram, is dependent upon the signal-to-noise ratio and upon the threshold setting. The time interval histogram has a gain setting which, if set too high, will cause broadening of the spectral display because of amplification of background noise. In the ATL system, the analogue velocity signal is the integrated average of the time interval histogram. Therefore, if there is a flow disturbance which has no net average velocity toward or away from the transducer, the analogue signal will sit at zero. In the examples where there is a net velocity, there will be a deflection from zero on the analogue velocity output and the time interval histogram will show spectral broadening displaced from the zero baseline. In the recordings obtained, care was taken to ensure the signals were not contaminated by noise by carefully adjusting the gain and sensitivity so that the recorded displays only showed true flow information. In order to achieve this, the gain was increased until the output was triggered by noise, then decreased until triggering was no longer observed.

The analogue signal is derived from the time interval histogram by passing it through integrating low pass filters. It therefore has an inherent delay depending upon the filter time constant. This time constant has been adjusted to produce minimal reproducible delays (0·03 s). Selection of the filter time constant is a matter of compromise between minimising this delay and producing a smooth curve. The influence of the angle theta between the ultrasound beam and the direction of blood flow is minimised when the suprasternal approach to the aorta is used since, from that location, the beam of ultrasound is nearly parallel to the direction of the central axis of the aorta.

Since none of these limitations of the technique limits its use for pattern recognition of abnormal velocity curves, discussion will be limited to the relation between the recorded curves and the pathological process producing the abnormal flow velocity.

VALIDITY AND SIGNIFICANCE OF RECORDED CURVES: DIAGNOSTIC VALUE

(A) *Normal subjects*

In normal subjects, the Doppler flow velocity patterns from the aorta are strikingly similar to those

Table 5 *Correlations between pulsed Doppler technique and classical procedures for diagnosing and assessing severity of aortic valvular lesions*

Diagnosis	*No.*	*Positive diagnosis*		*Sensitivity (%)*	*Specificity (%)*	*Correct evaluation of grading (%)*
		Classical procedures	*Echo pulsed Doppler*			
AS	16	Traumatic 11	10	90	87 pattern 100 pattern + timing	81
		Non-traumatic 5	4	87		
AR	22	Traumatic 17	16	94	88	82
		Non-traumatic 5	3	86	90	
AS + AR		Traumatic				
AS	10	10	9	90		77
AR			8	80		50

AS, aortic stenosis; AR, aortic regurgitation; "traumatic procedures" refer to haemodynamic, angiographic, and/or surgical findings.

of volume flow recorded using surgically implanted electromagnetic flowmeters in dogs and man,[24 25] or of flow velocity recorded experimentally and clinically using the pressure gradient technique,[26] the hot film anemometer,[27] and the electromagnetic flow velocity catheter probe during standard cardiac catheterisation.[28] The velocity curves therefore have the same physiological validity and significance.[29] Small positive diastolic oscillations are visible on some of our curves. Since a reflection wave is improbable at this level, they may be the result of diastolic wall displacements or artefact.

Effect of velocity profiles. The normal aortic velocity profile is almost flat except for a marginal region close to the arterial wall.[30–32] The recorded velocity pattern, whatever the site of recording, is therefore representative of the overall aortic flow velocity. However, the velocity curve is not directly related to volume flow since the aortic diameter may vary in diameter by a significant amount throughout the cardiac cycle.[33 34] Study of instantaneous flow velocity curves throughout the cardiac cycle demonstrates the physiological occurrence of flow disturbances during deceleration,[30] or present for a short time at the peak of ejection mainly in the ascending aorta.[27 35] These findings obtained with thin film anemometers agree with our observation of a slight broadening on the descending limb of the spectral curve and minute irregularities of the systolic summit. Our measured timing intervals are in general agreement with published reports for ejection time; the Q-onset interval of the velocity curve reflects the internal pre-ejection period taking into account the delay in the analogue velocity output.[36] No significant delay in onset of the systolic wave is found in normal subjects between the ascending aorta and the arch of the aorta.

Aortic orifice. The two deflections with coincident high-pitched signals on the loudspeaker are related to opening and closing movements of the aortic cusps whose echoes are simultaneously displayed on the echocardiogram, as previously noted by Johnson.[11] The deflections usually show the same positive sign, suggesting that they are generated by the motion of two different cusps. Nothing other than a brief early systolic disturbance can be recorded at this level in physiological flow.

(B) *Patients with aortic valve disease*

The velocity patterns recorded with the pulse echo Doppler in patients with aortic valve disease are also similar to those recorded using other techniques, both in experimental[37] and human studies; for instance hot film anemometer measurements in man with aortic stenosis,[30 35] electromagnetic flowmeter measurements at the time of surgery,[6 38] and electromagnetic catheter tip measurements in animals with artificially induced aortic regurgitation.[39] The externally recorded velocity curves therefore faithfully represent aortic flow velocity patterns and have the same pathophysiological significance as far as diagnosis and clinical applications are concerned.

The systolic broadening of the spectral display consequent on the dispersion of the dots on the time interval histogram probably reflects the irregular velocities of red blood cells present in turbulence generated by stenosis. The saw-toothed indentations seen on the analogue velocity curve may be considered a more obvious visualisation of the irregular character of turbulent flow velocities. The diastolic reverse flow velocity in aortic regurgitation is the graphical representation of the reflux in the aorta. In these lesions, the peak velocity is not adequately represented whenever turbulent flow is present, either in the spectral or the analogue output and some artefacts may be present in the zero-crossing output because of its inherent non-linear properties.

Effect of velocity profiles. Instead of the nearly flat normal velocity profile, aortic stenosis is characterised by an irregular profile throughout the aortic cross-section, the highest velocities corresponding to the jet which is most commonly found near the anterior aortic wall.[30]

Effect of aortic diameter variation. The aortic cross-sectional diameter may vary by as much as 50 per cent during the cardiac cycle in cases of severe aortic regurgitation.[40] Consequently, the finding of a larger negative than positive velocity does not reflect the true volume flow in cases of gross regurgitation as previously stressed by Tunstall-Pedoe for the subclavian artery.[41]

AORTIC STENOSIS

(A) *Aortic velocity curves*

In 10 out of 11 patients, the characteristic abnormal systolic pattern was found, allowing a definite diagnosis to be established. This abnormality was also present in the patients with mild stenosis who were assessed purely by non-invasive procedures. The second striking feature seen in our recordings was the presence of different velocity patterns at the various sites along the aorta. The most abnormal velocity patterns were found in the samples close to the aortic orifice but they could persist for some distance downstream or be replaced by a normal or nearly normal pattern, either at the distal part of the ascending aorta or in the arch. The severity of the lesion, therefore, was not assessed purely on the existence of the abnormal "S" wave present in every

stenosis, but on the persistence of the abnormality downstream from the valve. With this type of assessment, we could get a fairly good idea of the severity of the stenosis, comparable with the findings of invasive procedures (Table 6). This grading is in agreement with what we know of the propagation of turbulence downstream of a stenosed aortic valve.[30 35] In 45 per cent of the patients, the diastolic part of the curve was abnormal, exhibiting chaotic irregularities particularly when the recordings showed the maximum systolic disturbances. These findings may well be related to the diastolic persistence of turbulence previously shown in some individual cases.[30 35] The timing features are shown in Table 4. One significant anomaly was apparent when comparison was made with other groups of patients (Fig. 12).

There was a significant delay in the onset of the velocity curve when recorded from the aortic arch compared with that recorded from the ascending aorta, even in patients with mild aortic stenosis. This delay was also present in some samples recorded from the ascending aorta. It is not related to the left bundle-branch block seen in two cases since it is additional to the delay in the onset of the earliest velocity curve recorded near the aortic valve. The possibility of a technical fault cannot be excluded, though a possible distortion of the analogue curve was confidently ruled out by the simultaneous check with the spectral curve recording. Further studies will be required to elucidate this finding, which is peculiar to aortic stenosis in our study. It is consistent with the finding of Johnson *et al.*[11] who found later turbulence in the aorta than close to the aortic valve. Though some patients had extreme prolongation of the ejection time, the mean value did not reach a significant level ($p < 0.02$) probably because of insufficient numbers of tracings, since these ejection times are severely abnormal when measured from pressure or carotid pulse traces.[42 43]

Table 6 *Correlations between left ventricular outflow tract turbulence detection and classical invasive procedures in pure aortic regurgitation (diagnosis and prognosis)*

Positive diagnosis			
Classical procedures	*Echo pulsed Doppler velocimeter*		*Correct evaluation of grading* (%)
	Sensitivity (%)	*Specificity* (%)	
17	94	80	70

Same abbreviations as in Table 5.

(B) *Aortic orifice examination*

Disturbance of the spectrum at the aortic valve is a useful sign of organic valve disease but is not specific for aortic stenosis and may be encountered with aortic regurgitation, especially when the cause is endocarditis with vegetations. Abnormality is not immediately evident in some cases and may be shown only by tilting the transducer from the optimal position for obtaining echo structure visualisation. Locating the disturbances with an eccentric location of the gate may be a useful clue to the presence of a biscuspid aortic valve (Fig. 8).

AORTIC REGURGITATION

(A) *Aortic velocity curves*

The velocity curves we recorded are comparable with the demonstration of aortic backflow in aortic regurgitation using angiographic techniques,[40] surgically implanted electric flowmeters,[6 38] and recordings from catheter-tip velocimeters.[39] The finding of negative diastolic velocity in the aortic flow pattern using pulsed Doppler appears to be a graphic representation of the regurgitant flow and confirms the diagnosis of aortic regurgitation. False positive results from the sample volume being placed close

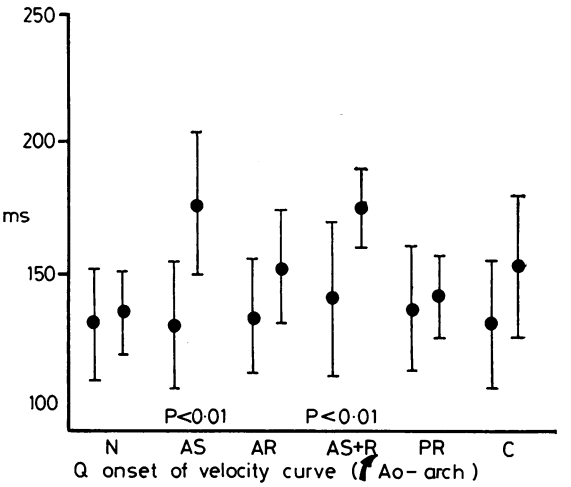


Fig. 12 *Comparative values of the Q-to-onset of the velocity curve time interval in the ascending aorta and in the arch (respectively, first and second vertical line for each group of subjects). ms, millisecond; N, normal subjects; AS, aortic stenosis; AR, aortic regurgitation; PR, patients with prosthesis; C, cardiac control group;* ● *mean value;* lower row, *the p values are indicated only when significant differences exist between the mean values. This diagram clearly shows the significant delay in the onset of the velocity curve at the aortic arch in patients with organic aortic stenosis.*

to the sinuses of Valsalva and recording discrete negative veolocities in diastole can be avoided by careful echocardiographic checking of the location of the sample volume. In view of the systolic changes in aortic diameter, it is clear that this qualitative method cannot be used to quantify the regurgitant flow and derive a regurgitant fraction. However, by using several different sample volumes at different sites in the aorta, it is possible to classify the regurgitation into three grades of severity. The possibility of either normal or disturbed systolic flow velocity which can be encountered in severe regurgitation[30] must be taken into consideration in assessing the severity of the lesion (Fig. 11).

Since there is no significant delay in the onset of the velocity curve at the arch in aortic regurgitation, this is a useful diagnostic criterion for distinguishing functional stenosis in severe aortic regurgitation from an organic valve stenosis, especially since the ejection time may be prolonged in both of these conditions. Very early onset of the systolic velocity curve may be noted in the ascending aorta in some cases where the end-diastolic ventricular and aortic pressures are almost equal. The insignificant delay in onset of the systolic curve compared with normal subjects could be a result of the frequent presence of aortic dilatation in aortic regurgitation.

(B) *Aortic valve orifice examination*

Systolic disturbances of the spectral display probably indicate functional stenosis though these were not always limited to early systole especially in patients with endocarditis. Diastolic turbulence is useful diagnostically but is less important in assessing prognosis.

AORTIC STENOSIS AND REGURGITATION

The velocity curves consistently showed anomalies characteristic of both stenosis and regurgitation though both anomalies were not evident on every sample. Sequential recordings along the aorta were required to confirm the diagnosis and make sure that one of the lesions was not being missed. The specific anomalies of timing were essential in assessing the presence of coincident aortic stenosis with significant aortic regurgitation.

AORTIC PROSTHESES

The study of these included two parts. (1) The recording of the velocity of opening and closing motion of the prosthetic valve: the amplitude and duration of these deflections were similar to those of normal aortic valves but were opposite in sign since they were related to the to-and-fro movement of a single element in the beam of the transducer. The polarity of the deflection varied according to the site of recording. No anomaly of these deflections was seen with a malfunctioning Starr-Edwards valve with paravalvular leak. Irregular amplitude of the deflections was noted with atrial fibrillation so this irregularity does not constitute by itself a sign of malfunction.

(2) Aortic velocity curve, with some spectral broadening in the time interval histogram and some saw-toothed indentations on the summit of the analogue velocity curve, is consistent with disturbed flow in the ascending aorta of patients with a prosthesis.[35] These patterns may be related to a pressure gradient across the prosthetic valve.[44 45] The absence of any timing abnormalities suggests the diagnosis of functional aortic stenosis. In diastole, the curve is similar to that of normal subjects except with a paravalvular leak when negative flow velocity was recorded as far distal as the aortic arch. These recordings led to the diagnosis of a paravalvular leak and, at operation, a paravalvular leak 1 cm^2 was found and repaired. It seems, therefore, that pulsed echo Doppler recordings may provide an additional method for assessing the function of valve prosthesis.

CONTROL GROUP

None of the characteristic abnormalities found in patients with aortic valve disease was encountered in this group. The curves were either identical to those found in normal subjects, or had a velocity pattern characteristic of the underlying condition, that is a systolic "S" wave of short duration in patients with severe mitral regurgitation, or traumatic ventricular septal defect or variable systolic amplitude in cases of atrial fibrillation with the amplitude of the "S" wave dependent on the preceding diastolic interval. There were minute negative diastolic oscillations where diastole was prolonged. However, in two cases of pulmonary stenosis, the "S" wave was indented but the normal timing of the velocity events ruled out aortic stenosis. These findings emphasise the importance of combining both pattern and timing informations. It seems likely that some aortic disturbances may occur in patients with pulmonary stenosis probably because of the proximity of the major vessels.[30]

Correlations with classical invasive procedures (Tables 5, 6)

PURE AORTIC STENOSIS

The Doppler method appears to be a useful diagnostic method, sensitivity and specificity being 87 to 90 per cent and reaching 100 per cent speci-

ficity when timing abnormalities were added to pattern recognition criteria. Assessment of the severity of the stenosis from the velocity curves was also reliable.

AORTIC REGURGITATION

The technique was equally valuable in the diagnosis and assessment of the severity of aortic regurgitation. Addition of the patients with mild regurgitation who had not been assessed invasively to the two groups hardly lessened the percentage of good correlations.

COMBINED STENOSIS AND REGURGITATION

The technique seemed to be equally valuable for diagnosis of patients with combined lesions. A correct evaluation of the grading of the stenosis was obtained in 77 per cent of cases, but there were some discrepancies in assessment of the severity of coincident aortic regurgitation, bringing the accuracy down to 50 per cent.

Discrepancies between Doppler assessment and invasive findings

DIAGNOSIS

False-negative diagnoses were exceptional and were made only in mild lesions (cases 10, 13, 30, 32, 38). They were all based on incomplete recordings. This underlines the critical need for a series of velocity samples to be obtained at different sites in the aorta for correct diagnosis. One false-positive diagnosis of aortic regurgitation resulted from placement of the sample volume close to the aortic wall. In the detection of turbulence, a high degree of sensitivity is preserved but specificity is less because of false-positive diagnoses in some cases of mitral regurgitation.

GRADING OF SEVERITY

Except for one case (case 36), discrepancies resulted from an overlap between grades. This was uncommon in pure lesions (cases 6, 27). In case 27, a low cardiac output might have led to erroneous underestimation. Though we have not experienced the same error with stenosis, the possibility must be borne in mind. The evaluation of combined lesions was satisfactory for the assessment of stenosis, with only two exceptions (cases 36, 38). Case 36 was the only one where there was a grading difference of 2: a tracing from a branch vessel was wrongly interpreted as an aortic arch recording. This mistake can be avoided by setting the sample volume at a suitable depth where it is unlikely to record from superficial vessels which do not always show the flow abnormalities. In combined lesions, assessment of aortic regurgitation by this technique was less satisfactory in this series, with four discrepancies (cases 29, 31, 36, 37). However, the differences involved only one grade.

Two of these discrepancies involved overestimation of the severity of the regurgitation. This probably resulted from measurements close to the jet from a stenotic valve where there may be large vortices with a negative component. To avoid this mistake we now record several samples close to each other in the same part of the aorta. In regurgitation the patterns are similar to each other and only alter progressively downstream, whereas with stenosis and its associated large vortices the pattern is chaotic. Where the results were discrepant, a low cardiac output has already been mentioned as a possible source of error and mitral valve lesions were often present in some cases. When aortic regurgitation is assessed purely on the detection of turbulence in the left ventricular outflow tract, the regurgitation may be wrongly estimated.

Limitations of method

Throughout this paper the technical limitations of the technique have been stressed. It is again emphasised that this is not a quantitative method of measuring flow or absolute velocity. From the clinical point of view, this is of minor importance since the diagnostic criteria are based purely on pattern recognition and timing abnormalities of the velocity curves. However, some rules have to be followed for accurate diagnoses using this technique. These include a careful echocardiographic check of the correct location of the Doppler sample, and the use of an adequate number of velocity samples which should be taken at a sufficient depth for the aortic samples to be well within the aorta. These velocity recordings are not always possible particularly in patients with emphysema. Rarely, in others, an adequate number of sites cannot be sampled for accurate assessment of the severity of the lesion, but, as a general rule, recording from the suprasternal notch is consistently successful. This is facilitated by the small size of the transducer. Deformity of the thoracic cage seems to be less of a drawback for this technique than for classical echocardiography. On the other hand, previous tracheotomy or presence of a tracheotomy tube makes the suprasternal approach extremely cumbersome.

Conclusions

Pulse echo Doppler blood velocity recordings need further technical improvements for a more complete demonstration of the complex flow disturbances generated in aortic valve disease. However,

for purely clinical purposes, such as diagnosis and assessment of the severity of the lesion, this technique is very useful and reliable, and is useful also for the assessment of prosthetic valve function.

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